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AN   2002:198848 CAPLUS
DN   136:321171
TI   Redox control of calcineurin by targeting the binuclear Fe2+-Zn2+ center at the enzyme active site
AU   Namgaladze, Dmitry; Hofer, H. Werner; Ullrich, Volker
CS   Mathematisch-Naturwissenschaftliche Sektion, Fachbereich Biologie, Universitat Konstanz, Konstanz, D-78457, Germany
SO   Journal of Biological Chemistry (2002), 277(8), 5962-5969
     CODEN: JBCHA3; ISSN: 0021-9258
PB   American Society for Biochemistry and Molecular Biology
DT   Journal
LA   English
AB   The interaction of protein serine/threonine phosphatase calcineurin (CaN) with superoxide and hydrogen peroxide was investigated. Superoxide specifically inhibited phosphatase activity of CaN toward RII (DLDVPIPGRFDRRVSVAAE) phosphopeptide in tissue and cell homogenates as well as the activity of the enzyme purified under reducing conditions. Hydrogen peroxide was an effective inhibitor of CaN at concns. several orders of magnitude higher than superoxide. Inhibition by superoxide was calcium/calmodulin-dependent. Nitric oxide (NO) antagonized superoxide action on CaN. We provide kinetic and spectroscopic evidence that native, catalytically active CaN has a Fe2+-Zn2+ binuclear center in its active site that is oxidized to Fe3+-Zn2+ by superoxide and hydrogen peroxide. This oxidn. is accompanied by a gain of manganese dependence of enzyme activity. CaN isolated by a conventional purifn. procedure was found in the oxidized, ferric enzyme form, and it became increasingly dependent on divalent cations. These results point to a complex redox regulation of CaN phosphatase activity by superoxide, which is modified by calcium, NO, and superoxide dismutase.
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DN   PREV200100574338

TI Nitric oxide synthase, **calcineurin**, and **superoxide dismutase** in cord and periphery-projection central neurons following axonal injury.
AU Tseng, G. F. (1); Tsai, S. Y. (1)
CS (1) Dept Anatomy and Cell Biology, Col Med, National Taiwan Univ, Taipei Taiwan
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 2036. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001
ISSN: 0190-5295.
DT Conference
LA English
SL English
AB We compared the responses of rat rubrospinal (RS) and facial motoneurons following axotomy. In the red nucleus (RN), dark nitric oxide synthase (NOS) immunoreactive cells started to increase 3 days, peaked 1-2 weeks, and decreased to baseline later following spinal tractotomy. Only lumbar-projection RS neurons were NOS(+) following upper cervical, but not lower thoracic tractotomy. Thus the proximity of axonal lesion to their cell bodies is critical to the elevation of NOS. At the same time, Mn and Cu/Zn-SOD remained unchanged in these neurons indicating the lack of protection of injured neurons since SOD can compete with NO for superoxide, thus reducing its neurotoxicity. Meanwhile calcineurin was moderately increased, which could upregulate NOS activity of axotomized RS neurons. In contrast, neurectomy resulted in an increase of Mn-SOD and a decrease of calcineurin in facial neurons, which also expressed high level of NOS. Thus, both cord and periphery-projection central neurons upregulated NOS expression following axonal injury, however, only the latter were protected from calcium-dependent NO production by downregulation of calcineurin and also from the formation of peroxynitrite by upregulation of Mn-SOD. These are likely to better protect periphery-projection central neurons from injury-induced reactive oxygen and/or nitrogen species toxicity. These differences and the fact that axotomized intrinsic central, but not periphery-projection neurons retain glutamatergic afferent synpases may be related to the inability of the latter to regenerate their axons and perhaps also to their degenerative fate following axonal injury.

L2 ANSWER 3 OF 12 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:503815 BIOSIS
DN PREV200100503815
TI Direct interaction between Cu,Zn-SOD and calcineurin.
AU Reuter, A. T. (1); Voelkel, H. (1)
CS (1) Dept Neurol, University of Ulm, Ulm Germany
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 873. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001
ISSN: 0190-5295.
DT Conference
LA English
SL English
AB Calcineurin is a calcium/calmodulin regulated phosphatase which has been implicated as a key player not only in the modulation of neurotransmission, but also in amyotrophic lateral sclerosis, ischemia/reperfusion injury and heart failure. Recently, it was shown that Cu,Zn **superoxide dismutase** protects **calcineurin** from oxidative inactivation and is copurified with calcineurin if using gel filtration. However, the mechanism of this protection and the nature of the association between SOD and calcineurin are still unknown. In this study, we show by means of protein chip technology, pull-down assays and immunoprecipitation that **calcineurin** can bind to **superoxide dismutase** directly.

L2 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2
AN 2001:179103 CAPLUS
DN 135:135873
TI A putative role for calcineurin in lymphopenia associated with chronic renal failure
AU Sasikala, M.; Sadasivudu, B.; Subramanyam, C.
CS Vijaya Diagnostic Centre, Osmania University, Hyderabad, 500 007, India
SO Clinical Biochemistry (2001), Volume Date 2000, 33(8), 691-694
CODEN: CLBIAS; ISSN: 0009-9120
PB Elsevier Science Inc.
DT Journal
LA English
AB One of the important features of chronic renal failure (CRF) is related to the assocd. hematol. disorders such as anemia, thrombocytopenia and lymphopenia. Because recent studies have identified calcineurin in serum, it was of interest to assay its activity in sera and lymphocytes in CRF to evaluate the relevance of calcineurin and reactive oxygen species (ROS) in the etiol. of lymphopenia that accompanies CRF. Malondialdehyde (MDA) levels were evaluated and the activities of **superoxide dismutase** (SOD) and **calcineurin** in sera and lymphocytes were assayed during progression of CRF. Data suggest that decreased calcineurin activity, concomitant with increases in ROS activity, in lymphocytes may account for the lymphopenia occurring during progression of CRF.
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L2 ANSWER 5 OF 12 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
AN 2001:555084 BIOSIS
DN PREV200100555084
TI Oxidative inactivation of **calcineurin** by Cu,Zn **superoxide dismutase** G93A, a mutant typical of familial amyotrophic lateral sclerosis.
AU Ferri, Alberto; Gabbianelli, Roberta; Casciati, Arianna; Celsi, Fulvio; Rotilio, Giuseppe; Carri, Maria Teresa (1)
CS (1) Dipartimento di Biologia, Universita di Roma 'Tor Vergata', Via della Ricerca Scientifica, 00133, Rome: carri@Bio.uniroma2.it Italy
SO Journal of Neurochemistry, (November, 2001) Vol. 79, No. 3, pp. 531-538. print.
ISSN: 0022-3042.
DT Article
LA English
SL English
AB Calcineurin is a serine/threonine phosphatase involved in a wide range of cellular responses to calcium mobilizing signals. Previous evidence supports the notion of the existence of a redox regulation of this enzyme, which might be relevant for neurodegenerative processes, where an imbalance between generation and removal of reactive oxygen species could occur. In a recent work, we have observed that calcineurin activity is depressed in two models for familial amyotrophic lateral sclerosis (FALS) associated with mutations of the antioxidant enzyme Cu,Zn superoxide dismutase (SOD1), namely in neuroblastoma cells expressing either SOD1 mutant G93A or mutant H46R and in brain areas from G93A transgenic mice. In this work we report that while wild-type SOD1 has a protective effect, calcineurin is oxidatively inactivated by mutant SOD1s in vitro; this inactivation is mediated by reactive oxygen species and can be reverted by addition of reducing agents. Furthermore, we show that calcineurin is sensitive to oxidation only when it is in an 'open', calcium-activated conformation, and that G93A-SOD1 must have its redox-active copper site available to substrates in order to exert its pro-oxidant properties on calcineurin. These findings demonstrate that both wild-type and mutant SOD1s can interfere directly with calcineurin activity and further support

the possibility of a relevant role for calcineurin-regulated biochemical pathways in the pathogenesis of FALS.

L2 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2002 ACS
AN 2001:611160 CAPLUS
DN 135:330058
TI Superoxide dismutase mutations of familial amyotrophic lateral sclerosis and the oxidative inactivation of calcineurin
AU Volkel, H.; Scholz, M.; Link, J.; Selzle, M.; Werner, P.; Tunnemann, R.; Jung, G.; Ludolph, A. C.; Reuter, A.
CS Departments of Neurology, O 25, University of Ulm, Ulm, 89081, Germany
SO FEBS Letters (2001), 503(2,3), 201-205
CODEN: FEBLAL; ISSN: 0014-5793
PB Elsevier Science B.V.
DT Journal
LA English
AB Approx. 10% of all familial cases of amyotrophic lateral sclerosis (fALS) are linked to mutations in the SOD1 gene, which encodes the copper/zinc superoxide dismutase (CuZnSOD). Recently, wild-type CuZnSOD was shown to protect calcineurin, a calcium/calmodulin-regulated phosphoprotein phosphatase, from inactivation by reactive oxygen species. We asked whether the protective effect of CuZnSOD on calcineurin is affected by mutations assocd. with fALS. For this, we monitored calcineurin activity in the presence of mutant and wild-type SOD. We found that the degree of protection against inactivation of calcineurin by different SOD mutants correlates with the severity of the phenotype assocd. with the different mutations, suggesting a potential role for calcineurin-SOD1 interaction in the etiol. of fALS.
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PB American Health Consultants, Inc.
DT Newsletter
LA English
WC 2130
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SO BIOWORLD Today, (11 Apr 2000) Vol. 11, No. 69.
PB American Health Consultants, Inc.
DT Newsletter
LA English
WC 2130

L2 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2002 ACS
AN 2000:84442 CAPLUS
DN 132:132319
TI A high throughput bioassay for screening for effectors of the interaction between **calcineurins** and Cu/Zn-**superoxide dismutases** for the identification of pharmaceuticals
IN Volkel, Helge
PA Germany

SO Eur. Pat. Appl., 16 pp.
CODEN: EPXXDW
DT Patent
LA German

Foreign Application - check for priority data

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 976823	A1	20000202	EP 1998-113876	19980722
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	WO 2000005363	A1	20000203	WO 1999-EP5220	19990722
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 9954124	A1	20000214	AU 1999-54124	19990722
	EP 1100912	A1	20010523	EP 1999-940032	19990722
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2002522015	T2	20020723	JP 2000-561309	19990722
PRAI	EP 1998-113876	A	19980722		
	WO 1999-EP5220	W	19990722		

AB Methods of screening for substances that affect the interaction between **calcineurins** and Cu/Zn-**superoxide dismutases** or the regulation of expression of their resp. genes are described. Such compds. may be useful in the treatment of neurol., immunol., or heart and circulatory diseases. Methods that use laser correlation spectroscopy to measure fluorescence-labeled proteins are described. The proteins can be labeled with fluorescent reporter groups or by fusing them with a green fluorescent protein or a novel fluorescent peptide substrate (RII-Fluophos).

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 4
AN 2000:514656 CAPLUS
DN 133:221124
TI Calcineurin activity is regulated both by redox compounds and by mutant familial amyotrophic lateral sclerosis-superoxide dismutase
AU Ferri, Alberto; Gabbianelli, Roberta; Casciati, Arianna; Paolucci, Egle; Rotilio, Giuseppe; Carri, Maria Teresa
CS Fondazione S. Lucia IRCCS, Universita di Roma "Tor Vergata,", Rome, 00133, Italy
SO Journal of Neurochemistry (2000), 75(2), 606-613
CODEN: JONRA9; ISSN: 0022-3042
PB Lippincott Williams & Wilkins
DT Journal
LA English
AB Calcineurin (CN) is a protein phosphatase involved in a wide range of cellular responses to calcium-mobilizing signals, and a role for this enzyme in neuropathol. has been postulated. We have investigated the possibility that redox modulation of CN activity is relevant to neuropathol. conditions where an imbalance in reactive oxygen species has been described. We have monitored CN activity in cultured human neuroblastoma SH-SY5Y cells and obtained evidence that CN activity is promoted by treatment with ascorbate or dithiothretiol and impaired by oxidative stress. Evidence for the existence of a redox regulation of this enzyme has been also obtained by overexpression of wild-type antioxidant Cu,Zn superoxide dismutase (SOD1) that promotes CN activity

and protects it from oxidative inactivation. On the contrary, overexpression of mutant SOD1s assocd. with familial amyotrophic lateral sclerosis (FALS) impairs CN activity both in transfected human neuroblastoma cell lines and in the motor cortex of brain from FALS-transgenic mice. These data suggest that CN might be a target in the pathogenesis of SOD1-linked FALS.

RE.CNT 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 11 OF 12 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 5
AN 1996:507216 BIOSIS
DN PREV199699229572
TI **Superoxide dismutase** protects **calcineurin** from inactivation.
AU Wang, Xutong; Culotta, Valeria C.; Klee, Claude B. (1)
CS (1) Lab. Biochem., Natl. Cancer Inst., NIH, Bethesda, MD 20892-4255 USA
SO Nature (London), (1996) Vol. 383, No. 6599, pp. 434-437.
ISSN: 0028-0836.
DT Article
LA English
AB Calcineurin is the only protein phosphatase known to be under the control of Ca-2+ and calmodulin. It is targeted by immunosuppressive drugs and has a critical role in T-cell activation. It is specifically inhibited by immunosuppressant immunophilin complexes, which enabled its function in regulating a wide range of cellular responses to Ca-2+-mobilizing signals to be identified. Calcineurin in situ is 10-20 times more active than in the purified form and is subject to a time- and Ca-2+/calmodulin-dependent reversible inactivation that is facilitated by small, heat-stable molecules. Here we identify a factor that prevents the inactivation of calcineurin in vitro and in vivo as the enzyme superoxide dismutase, which indicates that inactivation may be the result of oxidative damage to the Fe-Zn active centre of calcineurin. The redox state of iron provides a mechanism to regulate calcineurin activity by desensitizing the enzyme and coupling Ca-2+-dependent protein dephosphorylation to the redox state of the cell. The protection of **calcineurin** against inactivation by **superoxide dismutase** constitutes a new physiological role for this enzyme which enables the Ca-2+-dependent regulation of cellular processes to be modulated by the redox potential.

L2 ANSWER 12 OF 12 BIOCOMMERCE COPYRIGHT 2002 BioCommerce Data Ltd.
AN 0147430 BIOCOMMERCE FS Abstract
CO National Cancer Institute, USA (NCI) (1783), USA
Johns Hopkins University (1582), USA
SO Nature, 03 OCT 1996, vol. 3836599, Page(s) 434-437.
TC (General information not published in print edition)
AB NCI and Johns Hopkins University collaborators have found that **superoxide dismutase** (SOD) protects **calcineurin** from inactivation.

L Number	Hits	Search Text	DB	Time stamp
1	1	calcineurin near3 (superoxide adj dismutase)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/19 18:50